Headline Affluence putting health of Malaysians at risk
Date 23. Mar 2008
Media Title New Sunday Times
Section Focus
Circulation 171518
Readership 611000
Language ENGLISH
Page No 42,43
Article Size 524 cm2
Frequency Weekly
Color Full Color

YourHealth

Affluence putting health of Malaysians at risk

Prof Dr Ian Caterson says metabolic disease is slowly making its presence felt among children and adolescents

Malaysia is on track to becoming a developed nation — that is the good news. But the bad news is that Malaysians suffer from many diseases predominantly seen in fully developed countries, writes **ANNIE FREEDA CRUEZ**

BLAME it on metabolic disorders. Health Ministry statistics show that diabetes is today a common Malaysian malaise while coronary artery disease and obesity rates are reaching alarming levels, even among the young.

These diseases are all mostly the result of metabolic disorders caused by abnormal chemical reactions in the body that disrupt the metabolic process, resulting in too much of some substances or too little of others that one needs to stay healthy.

These diseases have been blamed on the increasing affluence of Malaysians, where they consume more rich food, indulge in sinful snacks and exercise less due to time spent at work.

Chronic metabolic disease, which includes coronary heart disease, hypertension, dyslipidaemia and impaired glucose metabolism, is already the dominant topic in healthcare in many parts of the world.

Medical experts are predicting that over the next 20 years the problems caused by this cluster of disorders are expected to increase.

University of Sydney's School of Molecular and Microbial Biosciences head, Professor Dr Ian Caterson, said this was particularly true in Asia, where these problems were increasing exponentially.

"What is often forgotten is the prevention and treatment of one of the major underlying causes of these chronic metabolic diseases — obesity, and particularly abdominal obesity," said Dr Caterson, who has over 170 publications in scientific journals and books.

Dr Caterson, who is also Boden Professor of Human Nutrition at Sydney University, said visceral fat releases hormones and inflammatory factors which cause or exacerbate these problems.

Reducing abdominal fat needs to be one of the priorities of prevention and of management, said Dr Caterson, who will be one of the speakers at the four-day conference on metabolic disorders in Kuala Lumpur.

He said the problem of insulin resistance and abdominal adiposity in Asia was worse than in other parts of the world.

He said the best way to reduce abdominal adiposity was by lifestyle change and where necessary, the use of weight loss pharmacotherapy or other adjunctive treatment.

There are several drugs available, those centrally acting (rimonabant and sibutramine) and peripherally acting (orlistat).

"It is interesting to note that the centrally acting drugs, as well as reducing abdominal adiposity, increase HDL cholesterol as well.

"Rimonabant may have specific effects on insulin resistance and diabetes beyond that produced by the degree of weight loss alone and this may be due to changes in adiponectin."

While lifestyle change may appear difficult, there are basic approaches that can bring about change, including:

- **Blood pressure control:** Weight loss helps but additional treatment may be necessary.
- **Lowering of cholesterol:** Again, weight loss and diet are important but the addition of specific drugs (particularly statins in those with diabetes) must be considered.
- **Diabetes control:** It is often forgotten that weight loss, especially abdominal fat loss, can help to control diabetes and

CONTINUED NEXT PAGE

FROM PAGE 42

impaired glucose metabolism. Use drugs that do not cause excessive weight gain and go to other treatments as necessary.

Dr Caterson said the problem with metabolic disorders was that there were a lot of individuals with them who, while getting better at tackling the problems, were not good at preventing them or even recognising the early stages.

"We recognise that increased adiposity, particularly abdominal adiposity, seems to have an underlying role.

"However, the cause of the increased adiposity is not well understood; there is genetic predisposition, epigenetic change, and environmental and social change (basically producing changes in food intake and in activity), but there is not a single pattern that can explain all cases."

Rather, there are multiple interlinking causes. This makes early intervention and/or prevention difficult but not impossible.

To intervene, Dr Caterson said, doctors needed to recognise those at risk.

"It is not difficult to assess adiposity clinically but yet we don't. Even if we do, we find it difficult to broach the subject with patients."

He pointed out that metabolic disease had started and was slowly making its presence felt among children and adolescents.

"We are getting better at managing the crises produced by metabolic disease but individuals still suffer and their quality of life reduced. Even those who are "just" obese have significantly reduced quality of life."

■ **A four-day conference on metabolic disorders themed "Integrative Approach to Healthy Living" will start at Berjaya Times Square Conference Centre in Kuala Lumpur on Thursday. The conference, organised by the Health Ministry, Universiti Putra Malaysia and the Malaysian Healthy Ageing Society, deals with diagnosing and managing metabolic disorders.**

Headline **Affluence putting health of Malaysians at risk**
Date **23. Mar 2008**
Media Title **New Sunday Times**

Metabolism is the process your body makes energy from the food you eat. Food is made up of proteins, carbohydrates and fats. Chemicals in your digestive system break the food parts down into sugars and acids, which are the body's fuel.

Your body can use this fuel right away, or it can store the energy in body tissues, such as in the liver, muscles and body fat. A metabolic disorder occurs when some organs, such as the liver or pancreas, become diseased or do not function normally. An example is diabetes.

THE GOOD & THE BAD

CHOLESTEROL can't dissolve in the blood. It has to be transported to and from the cells by carriers called lipoproteins.

Low-density lipoprotein, or LDL, is known as "bad" cholesterol. High-density lipoprotein, or HDL, is known as "good" cholesterol.

These two, together with triglycerides and Lp(a) cholesterol, make up the total body cholesterol count, which can be determined through a blood test.

LDL cholesterol: When too much LDL (bad) cholesterol circulates in the blood, it can slowly build up in the inner walls of the arteries that feed the heart and brain.

Together with other substances, it can form plaque, a thick, hard deposit that can narrow the arteries and make them less flexible. This condition is known as atherosclerosis. If a clot forms and blocks a narrowed artery, heart attack or stroke can result.

HDL cholesterol: About one-fourth to one-third of blood cholesterol is carried by high-density lipoprotein (HDL). It is known as "good" cholesterol because high levels of HDL seem to protect against heart attack. Low levels of HDL (less than 40 mg/dL) also increase the risk of heart disease.

Medical experts think that HDL tends to carry cholesterol away from the arteries and back to the liver, where it's passed from the body. Some experts believe that that HDL removes excess cholesterol from arterial plaque, thus slowing its buildup.

Triglycerides: Triglyceride is a form of fat made in the body. Elevated level of triglycerides can be due to overweight/obesity, physical inactivity, cigarette smoking, excess alcohol consumption and a diet very high in carbohydrates (60 per cent of total calories or more).

People with high triglycerides often have a high total cholesterol level, including a high LDL level and a low HDL level.

Many people with heart disease and/or diabetes also have high triglyceride levels.

Lp(a) cholesterol: Lp(a) is a genetic variation of LDL cholesterol. A high level of Lp(a) is a significant risk factor for the premature development of fatty deposits in arteries.

Lp(a) isn't fully understood, but it may interact with substances found in artery walls and contribute to the buildup of fatty deposits.

■ **Source: MedlinePlus**